A lack of insight? — Understanding some ways inadvertent iatrogenic harm can occur (part 2)

To briefly recap Part 1: I tried to show how there could be an escalation in the severity of someone's mental health presentation:

- from initially presenting with some mild mental health issue, and receiving a diagnosis and a prescription
- to potentially experiencing adverse effects from the prescribed drug
- effects which are then often mistaken for symptoms of some underlying condition
- then receiving consequently a more serious diagnosis and being prescribed other psychiatric drugs
- which could result in cognitive impairments or akathisia
- potentially leading to, additional diagnoses and a poorer prognosis; or the patient attempting to come off their psychiatric meds; or them feeling a need to 'self-medicate'
- hence to complications from substance use and/or other psychiatric drugs, or their withdrawal effects
- frequently resulting in what are perceived as 'relapses' and to the person becoming a "frequent flyer" for services.

'Epistemic Injustice'

So, what happens when people try to speak about adverse drug and drug withdrawal effects to doctors?

All too often they have not been believed, their claims written off as nonsense or delusion. Some reasons for this are:

- The doctors get information about the drugs from the pharma companies, either directly or indirectly. Adverse effects (including withdrawal syndromes) will tend to be glossed over in pharma's presentations.
- Prescribers probably won't be comfortable admitting to themselves, let alone anybody else, that their treatment decisions have been harmful
- Lack of guidance of psychiatric drug withdrawal from professional bodies — only beginning to change recently.
- A lot of doctors will be too busy in daily practice to keep up to date with latest research and so on.
- The power imbalance between an authoritative psychiatrist and a troubled psychiatric patient makes its easy to write off the latter's complaints as simply being manifestations of mental illness.

Patients experiencing adverse effects from treatment will be viewed through a prism of psychiatric characterisations; potentially a *weaponised vocabulary*. It's easy for professionals to dismiss a patient's reports. As having a psychiatric diagnosis and history means they can be deemed inherently unreliable as a self-witness.

They've got something wrong with them!

Breaking down psychiatric diagnoses

A look at some common assumptions about psychiatric diagnoses.

My sense of what's implied by the uncritical use of language by staff in services, and in the general public; when they say, for instance, '*he's got schizo..'* or '*she's got bipolar'* is that there is effectively a misapprehension of the nature of psychiatric diagnostics. That moves over them as theoretical constructs, to the assumption that they pick out some inherent feature of the patient's biological makeup. Explicitly or otherwise, the tendency is to **reify** some diagnostic construction into a real concrete property, that people with the diagnosis all allegedly have inside them.

Such ideas stem, in large part, from a psychiatric paradigm that had taken hold of the profession by the 1990's and are still, I'd say, reflected in guidelines, training, as well as public attitudes.

"The major psychiatric illnesses are diseases. They should be considered medical illnesses, just like diabetes..." Nancy Andreasen 1984

Diagnostics considered in the abstract:

- Taking a cluster of symptoms that often seem to present together
- Producing, effectively, some checklist of symptoms (S_1, S_2, S_3…), perhaps, with some heuristic for differentiation from other sets of symptoms
- This set is given a name — ‘X’

Then proceeding, implicitly, to the claim that

- Patients exhibit those symptoms because they ‘have X’.

This is clearly vacuous. We might, for example, enquire along these lines:

Q. Why is the patient delusional?

A. They’re delusional because because they’ve got schizophrenia.

Q. How do we know they’ve got schizophrenia?

A. Well, because they’re delusional.

And, we won’t have really explained or added anything.

Most philosophers of science, following Karl Popper, would say that non-falsifiable claims are not scientifically valid claims.

A diagnostic construction may have some legitimacy perhaps, as a *categorisation*, some marker in the conceptual terrain. However, like the idea of the ‘average voter’ it has been abstracted away from the unique makeup and life circumstances of people. It picks out certain features of interest and ignores all the rest.

A categorisation is not an explanation, much less a causal attribution.

The usually tacit assumption is that the diagnosis has a one-to-one mapping onto some *in principle* identifiable feature in the patient’s biological makeup. Even though its specific nature can’t be explicitly spelt out to patients and families, and there is no lab test that could confirm it. It’s taken as read that there will be some such identifiable common pathology for the diagnosis.

Typically, if a patient has more than one psychiatric diagnosis, then they are taken as having as many separate (discrete) psychiatric conditions. With the distinct diagnoses implying discrete diseases. *Some patients and families do identify with psychiatric diagnoses, feeling that they have as many separate illnesses as they have diagnoses.*

I want to say that, in such cases, they may be suffering from a nervous system dysregulation, which may well be complex in causation, but whose occurrence need not be attributed to the co-presence of discrete illnesses.

Not least, when their aetiology remains mysterious. Attempts to find, for example, genetic markers for ‘schizophrenia’ have failed. Where there may be a link to ‘copy number variants’, this perhaps applies to at most 3% of those with the diagnosis.

Some ideas for thinking about causation

Do people know the plot of Murder on the Orient Express?

[Spoiler]

After interviewing each of the remaining passengers on the train carriage, Poirot - in some trance-like state — realises that that all of them are responsible. Each of them had been affected by a horrific crime the victim had gotten away with. So they agreed together to carry out his killing as a joint act. So that responsibility for the murder wouldn't lie with any single one of them.

And, in looking to explain 'psychosis', or 'bipolar' etc, it's probably mistaken to assume that there must be *a single culprit*, or any single cause that accounts for the condition.

Complex causation & multifactoriality

Well, the Orient Express story is obviously not the most appropriate model, as the victim's death is really 'over-determined'. And generally, it's not good in a theory of causation for the effect to be overdetermined.

It occurred to me that a **dam being breached** might be a better analogy. *There is, say, some confluence of causes: eg, heavy rains over the tributaries of the river upstream, after recent extensive deforestation; so more water flows downstream and faster; the spillway channel around the dam is blocked up; and the sluice gates haven't been maintained properly, and so on.*

Illustrative image. One advantage of a dam analogy is that it fits with schematic talk of upstream and downstream causes and effects, sometimes employed in neuroscientific models. (Such as Oliver D Howes -Dopamine Hypothesis of Schizophrenia: version III the final common pathway)

Any one factor by itself wouldn't be sufficient, but their all occurring together is enough for the dam to be breached. This is basically a 'stress-vulnerability model', but I hope it illustrates the point that there are often several contributory factors to take into account. And that within these factors the widest context of environmental, social and psychological influences can play a role in shaping what might happen at the (presumed neurophysiological level) focal point.

A model of multiple contributory factors had been put forward by Dale Bredesen MD in relation to Alzheimers disease. Following his reasoning, many contributory factors mean that treatment interventions should have many targets.

He argues that the amyloid plaques, targeted in current drug treatments for Alzheimers, can be understood as a defence mechanism of the body; being more like a symptom rather than the pathology in itself. According to the view, just targeting that symptom does little to address its underlying causes.

Certain interest groups object to this approach on the grounds that it doesn't identify and target a *'common underlying neurophsyiological pathology'*. But the nub here, is whether its valid to assume that there has to be such a *'common underlying pathology'* to target. And, even if there is, it may arise from potentially different sets of causes.

I think an idea from philosophy of science and mind is also relevant here. The idea of ‘multiple realizability’ posits that some ‘higher level’ phenomenon, such as consciousness, could potentially be realized or instantiated by differing arrangements of ‘lower-level’ physical properties.

Descriptions versus explanations

If, for the sake of argument, we were to accept that psychosis only happens with *elevated dopamine synthesis*; this would not necessarily mean we have a good explanation for psychosis.

To make another analogy, if say, we want to know:

Why is the water in the kettle boiling?

It may help to have a microphysical description of an ensemble of H_20 molecules gaining sufficient kinetic energy to break their inter-molecular bonds, and so on. But the type of explanation we may be more interested is *eg*, that *Mrs Miggins wants a cup of tea!*

The micro-level description is useful to an extent, but it is not necessarily capturing the most pertinent causal origin.

Allowing that potentially different factors can result in the type of ‘dysfunction’ of interest; we can combine the idea that similar symptoms (as mental kinds) can potentially arise from distinct sets of causes, with the neuroscientific analogy of ‘upstream’ tributaries and a ‘downstream’ main channel. Implying that there may well be more than one way to address the ‘dysfunction’ and moreover, that the drastic option, of intervening directly on the neurophysiological main channel *as it were*, need not be called for.

Dispensing with a disease model

20% experience paranoia type complaints

In recent years there has been increasing recognition that the type of symptoms associated with the diagnosis can be quite common. And its heterogeneity suggests that it might be better understood as an ‘umbrella term’ rather than as a specific illness.

How can something be common place, yet non-existent?

Well, you see Santa Claus in lots of different places each year, and millions of presents appear overnight in children's bedrooms. But for all that, there is actually no such single entity responsible (the *role* of Santa Claus looks to be *multiply realizable*). And I'd suggest that explaining millions of individual episodes of psychosis, by recourse to a mysterious entity like 'schizophrenia' involves similar 'magical thinking' - although far less benignly.

Institutional inertia

Although more nuanced theoretical approaches have been put forward by leading experts, it seems this has not translated into wider changes in standard clinical practice.

At the end of Murder on the Orient Express, the decision makers on the train aren't willing to allow the consequences of Poirot's complex theory; implying a group of respectable people carrying out a murder together. They instead prefer to inform the authorities on the basis that some mysterious killer must have snuck onto the train and then off again.

It seems to me that, despite paying lip service to the many factors that can contribute to mental health and ill health, services at the end of the day, default to the position that a person's issues are basically caused by some single illness - mysterious as it is.

Diagnostic Overshadowing

This unreflective construal — of a provisional categorisation as an actual disease— may allow that medics stop looking for the real causes. Assuming that they already know what the issue is and how it should be addressed. So, they stop looking - *feeling confident that the patient has a diagnosis, and there are already established guidelines to follow for that* - rather than make a difficult adaption to dealing with a complex reality. Automatically jumping to

conclusions in that way can mean unnecessary treatment and unnecessary harm. As happened in this unfortunate young lady's case:

Having cerebral symptoms due to a rare brain disease, her case was once taken as 'treatment resistant schizophrenia'. Being labelled with that will mean getting *drugged up to the eyeballs with neuroleptics. W*hich would not have done Daisy's condition, or her quality of life any good.

She argues, I think fairly, that she didn't get the right right tests and checks when she should've got them, because her issues were written-off under different psychiatric diagnoses. Which resulted in undue delay in her actual brain disease being properly diagnosed.

Overmedicalisation?

I discussed the 'overmedicalisation' of mental health a bit earlier. But this looks to me to be a case of 'undermedicalisation'. I'd argue that it's the amorphous nature of diagnostic concepts like 'schizophrenia' and the seemingly unfalsifiable ascription of them to patients, that can allow misdiagnoses such as Daisy's to occur.

People in the survivor and critical psychiatry movements often talk about the 'overmedicalisation' of mental health. But I think it's worth exploring, a bit more, what people mean when they say "psychiatry is over-medicalised". I

think this is in some ways a bit of a misnomer - or a misapprehension.

Historians of psychiatry write that in the latter part of the 20th century the profession tried to disassociate itself from Freudian psychoanalysis. In doing so it tried to lay claim to a scientific legitimacy that it didn't really possess.

As far as I understand it, much of the logic underlying these pretensions to medical legitimacy was derived from the mechanisms of action of certain drugs. Drugs whose efficacy in treating certain symptoms had been discovered by accident. Joanna Moncrieff has emphasised that we should understand any efficacy of psychiatric treatment through a 'drug-centred model' rather than from a 'disease-centred model'.

Let's breakdown what we might mean by saying psychiatry is over-medicalised. *Are we saying that:*

- A medical approach is simply not appropriate

or — as psychiatrists like Niall McClaren and Peter Breggin have done — *are we saying that:*

- That the biomedical model of psychiatry doesn't really constitute or add up to being a medical discipline worthy of the name.

To me, the unreliability of diagnoses and the wide variety in their course and outcome, shows there is not really much depth to psychiatry as a medicalised paradigm. When anecdotally, for instance, attempting to find a particular antipsychotic that the patient tolerates relatively well is basically guesswork and trial and error.

Whilst accepting that, for many people, mental health issues, originating primarily from psychosocial factors, have been overmedicalised. I think it is also fair to say, there is probably a significant minority that may present due, partially at least, to causes that are within the legitimate preserve of medical science (for example, the connection to auto-immune encephalitis in some diagnosed with 'schizophrenia'). And that people falling in this group may receive care that can be said to be 'undermedicalised'.

I would stress again the importance of being as open-minded as possible about the range of potential causes for the clinical symptoms of interest: environmental, social, lifestyle, nurture — all of which can influence both our psychology and physiology, and the interplay between them.

A Before and After

Moreover, as we discussed earlier in relation to 'antipsychotics', there is a *before and after* with psychiatric drugs. A complicated prescribing history is likely to mean greater propensity to issues like *kindling* and *sensitization.* Where sometimes seemingly minor changes in levels of psychotropic compounds, or even changes in the patient's environment can precipitate crises.

These consequences of treatment then complicate the picture. This necessitates different sets of recommendations for drug naive first episode patients, as opposed to people who've already been exposed to neuroleptics or anti-convulsants, etc. The non-drug naive group needing support with switching, reducing or discontinuing meds safely, due to potential complications like supersensitivity and kindling.

David Healy has proposed that these type of effects be understood as amounting to a "drug dysregulation syndrome". The issues can be long-lasting and require ongoing efforts from those affected, in order to mitigate their impact on daily life.

As I've tried to show, the apparent clinical presentation of those effects will tend to overlap with the symptoms associated with certain psychiatric diagnoses. Consequently, these complications have often been mistaken by clinicians as evidence of a worsening psychiatric illness. There has been much resistance in the professional to entertaining this as a possibility.

Food for thought

The foregoing has tried to scratch the surface of how things can go wrong in mental health treatment. I'll try to end by pointing, quickly to ideas on what should be done instead. I'm really thinking more here about what can be done to further *prevention* and *sustaining recovery*, rather than about crisis interventions.

Are processed foods, sometimes with addictive properties, contributing to the ‘mental health crisis’?

Some researchers have suggested fructose (as found in corn syrup) looks to be one potential causal contributory factor to manic and ADHD type behaviours.

I’m not saying that’s the answer. But why would not tick it off the list of potential factors that we try to address? Before *eg,* prescribing amphetamines to youngsters. Similarly, in responding to relatively mild depression or anxiety like complaints, *why not examine diet, lifestyle, thinking styles before*

resorting to SSRIs?

Such diet and lifestyle interventions might benefit some people and come at low risk, even if asking for small sacrifices from people. I've suggested previously that we should be looking to help more susceptible people through a range of *soft* interventions.

Relevant reading

- *Kindling in psychopharmacology: Unveiling an overlooked clinical challenge with significant implications* — Laura Guerra, et al
- *Interoception or Neuroplasticity* — David Healy — (exploring what can be done to mitigate drug evoked nervous system dysregulation)